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The hemodynamics of tracheal intubation in critically ill patients: a narrative review

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Abstract

Background Tracheal intubation is a ubiquitous but high-risk procedure in critically ill patients, often required in the setting of severe hypoxemia, shock, or metabolic acidosis. Despite being a life-saving procedure, it is associated with a high incidence of peri-intubation adverse events. In a large international study (INTUBE), more than 40% of intubations in critically ill patients were complicated by cardiovascular instability, approximately 10% by severe hypoxemia, and over 3% by cardiac arrest, highlighting the magnitude of risk in this population. Among these complications, cardiovascular instability—particularly hypotension—emerges as the most frequent and most consequential, being independently associated with increased intensive care unit (ICU) and 28-day mortality. However, robust randomized evidence to guide optimal peri-intubation hemodynamic management remains scarce, leaving clinicians to rely largely on physiologic reasoning.

Main body Hemodynamic physiology during intubation is highly dynamic and influenced by cumulative and interacting threats. Critically ill patients often present with an endogenous catecholamine surge that temporarily preserves perfusion but reflects a fragile reserve. The administration of induction agents rapidly abolishes this sympathetic drive, leading to abrupt decreases in systemic vascular resistance and cardiac output. Apnea introduces hypoxemia, hypercapnia, and acidosis, all of which further impair myocardial contractility and catecholaminergic response, with increased arrhythmogenic risk. The subsequent initiation of positive-pressure ventilation raises intrathoracic pressure, reduces venous return, and increases right ventricular afterload, especially in the context of acute respiratory distress syndrome with hypoxic pulmonary vasoconstriction or pulmonary microthrombosis. Finally, post-intubation sedation, ventilator settings, and vasopressor therapy continue to shape the hemodynamic trajectory.

Conclusions This narrative review synthesizes current evidence and physiological insights into a structured timeline extending from induction to post-intubation care.

Keywords Propofol, Ketamine, Etomidate, Intubation, Hypotension, Intensive care units

Introduction

Tracheal intubation is a routine but high-risk intervention in critically ill patients, who often present with physiologic derangements, such as hypovolemia, vasoplegia, hypoxemia, and acidosis [1]. These conditions substantially increase the risk of hemodynamic collapse during airway management. In the large international cohort of the INTUBE study, 42.6% of ICU intubations were complicated by cardiovascular instability, 9.3% by severe hypoxemia, and 3.1% by cardiac arrest [2]. Among these, intubation-related hypotension—the most common

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complication—was associated with significantly higher ICU and 28-day mortality [3]. These findings underscore both the high incidence and the detrimental impact of peri-intubation hypotension, even when transient.

The concept of a *physiologically difficult* airway refers to patients who are particularly vulnerable to adverse physiological changes during intubation driving peri-procedural complications [1]. Unlike an anatomically difficult airway, which involves challenges in visualizing or accessing the airway, a physiologically difficult airway reflects underlying cardiopulmonary vulnerability. The two conditions can overlap and mutually influence: successful management of an anatomically difficult airway may help avert subsequent physiological deterioration as first-pass intubation failure has been associated with an increased risk of adverse events. A clear understanding of the dynamic physiologic changes surrounding intubation is, therefore, essential.

Given the high frequency of cardiovascular instability during intubation in patients with physiologically difficult airways, this review focuses on the fundamentals of hemodynamic physiology. Specifically, we discuss how hemodynamic status evolves during intubation, with particular attention to loss of sympathetic tone after induction and the transition to positive-pressure ventilation. Finally, we propose a practical, stepwise peri-intubation care bundle designed to address these physiological alterations. We aim to provide a practical framework for anticipating hemodynamic alterations, identifying critical points of vulnerability, and implementing targeted preventive strategies. By integrating pathophysiological mechanisms with bedside considerations, we highlight opportunities to reduce the incidence of cardiovascular collapse and improve outcomes in this high-risk patient population.

Time course of hemodynamic changes during intubation

Hemodynamic instability during tracheal intubation is not the result of a single event but of several predictable processes that unfold in sequence. Viewing this as a timeline—from pre-induction through induction, apnea, initiation of positive-pressure ventilation, and into post-intubation care—helps clinicians anticipate risk and target preventive strategies. Patients often enter the procedure in a fragile equilibrium. Apparent stability may be maintained by high sympathetic drive, which reflects limited physiologic reserve rather than true cardiovascular robustness. The administration of induction agents then disrupts this balance, rapidly blunting adrenergic tone and exposing hidden circulatory vulnerability.

The onset of apnea adds another layer of stress. Desaturation, hypercapnia, and resultant acidosis develop quickly. This cumulative burden sets the stage for deterioration. With the initiation of positive-pressure ventilation, intrathoracic pressure rises abruptly, reducing venous return and altering ventricular loading. In compromised patients, these changes can convert vulnerability into overt cardiovascular collapse. Even after the endotracheal tube is secured, hemodynamic risk persists. Ventilator settings, residual drug effects, and evolving physiology may either stabilize the patient or precipitate secondary deterioration.

Across all these phases, the threats—drug effects, apnea, ventilatory pressure shifts, and loss of sympathetic drive—are cumulative rather than isolated. Recognizing intubation as a trajectory instead of a single moment underscores the importance of continuous anticipation and proactive management (Fig. 1).

Adrenergic collapse during induction: endogenous and exogenous factors

Hemodynamic implications of catecholamine surge prior to induction

In patients with shock, the release of endogenous catecholamines—primarily epinephrine and norepinephrine—serves as a compensatory mechanism to maintain cardiac output and systemic vascular resistance [4]. Moreover, conditions such as pain (e.g., in trauma patients or during procedures, such as arterial cannulation), agitation or fear may further increase the adrenergic response of critically ill patients undergoing intubation.

However, excessive levels of circulating catecholamines may increase myocardial oxygen consumption, exacerbate ischemia, and precipitate cardiac arrhythmias [5]. In the INTUBE study, lower systolic blood pressure and elevated heart rate prior to intubation were independently associated with an increased risk of cardiovascular instability during the peri-intubation period [2]. These findings suggest that such patients may already be functioning at the limits of their compensatory capacity. Even minimal reductions in sympathetic tone—such as those induced by anesthesia—can trigger cardiovascular collapse in this vulnerable population [6, 7]. Clinicians should interpret this physiological state as a warning sign of impending instability rather than reassurance of hemodynamic adequacy. Early recognition of a catecholamine surge and timely hemodynamic optimization may mitigate the risk of collapse during induction. This physiological concept has direct clinical relevance, as later confirmed by large observational cohorts.

In the operating room, anesthesiologists often view the adrenergic surge accompanying laryngoscopy as harmful, since it can precipitate hypertension, tachyarrhythmias, and myocardial ischemia in otherwise stable surgical patients [8, 9]. Blunting this sympathetic response with

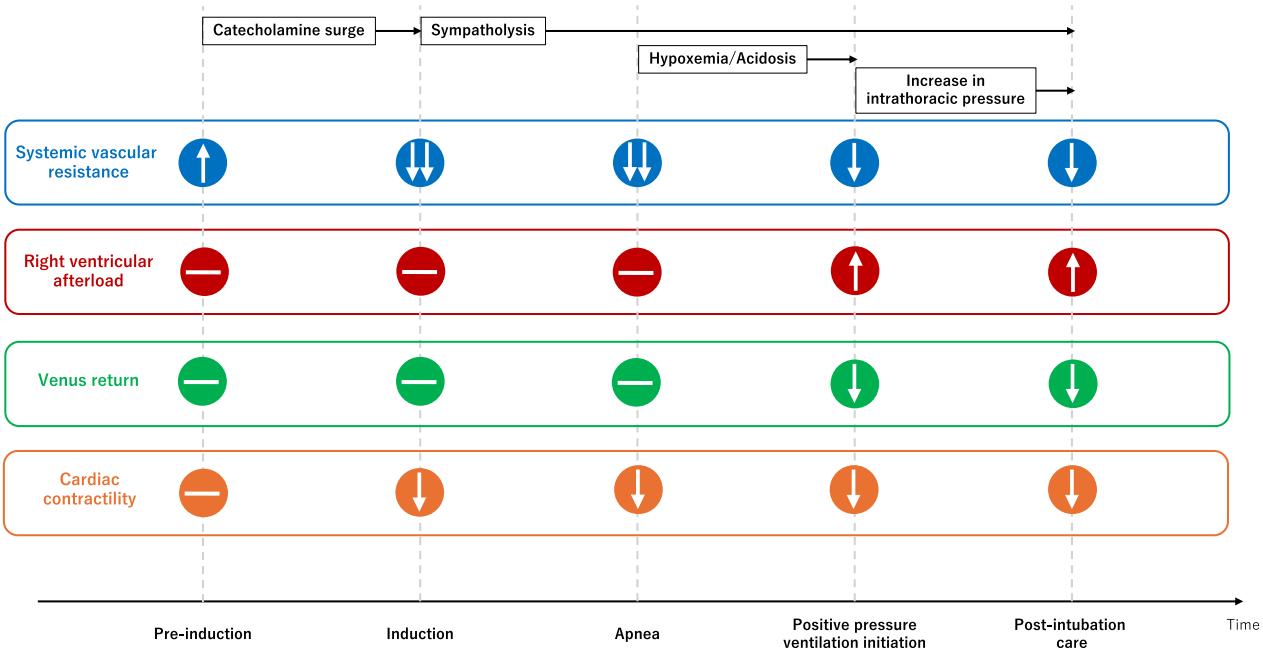

Fig. 1 Conceptual hemodynamic timeline of tracheal intubation in critically ill patients

opioids, beta-blockers, or deeper anesthesia is, therefore, a routine goal. By contrast, in the ICU, patients frequently depend on this adrenergic overdrive to sustain perfusion. In such settings, blunting the response—whether by induction drugs, analgesics, or simply relieving pain and agitation—can remove the final layer of circulatory support and precipitate collapse [10]. This contrast highlights a crucial physiological difference, i.e., what is protective in the operating theater may be catastrophic in the ICU.

The reliance on a catecholamine surge should, therefore, be recognized as a marker of circulatory fragility [11]. Elevated heart rate or stress-induced hypertension at the bedside should not be interpreted as stability but as signs of adrenergic dependence. Recognizing this physiology emphasizes the need for proactive hemodynamic preparation before induction.

Sympatholytic effects of induction agents

Induction agents play a crucial role in achieving amnesia, suppressing discomfort and agitation, and facilitating tracheal intubation, particularly in critically ill patients [12]. However, these agents universally blunt sympathetic tone, thereby increasing the risk of cardiovascular collapse [13]. Importantly, patients with apparently normal or even elevated pre-induction blood pressure may still experience rapid hemodynamic deterioration once sedation is initiated, as their normotension may reflect a stress-induced catecholaminergic state rather than true cardiovascular stability.

When rapid sequence induction is applied, the sympatholytic effects occur abruptly, often within less than a minute. In such cases, reactive hemodynamic interventions in response to overt hypotension may be too late to prevent organ hypoperfusion. Clinicians should then anticipate the loss of sympathetic tone following induction, which typically precipitates a marked drop in blood pressure and cardiac output. Preventive strategies aimed at minimizing the occurrence and duration of hypotension are essential to maintaining perfusion during this critical period.

Pharmacological effects of induction agents on the hemodynamics

Among induction agents, propofol remains widely used for its rapid onset and reproducible pharmacokinetic properties [14], but it is consistently associated with hypotension due to myocardial depression and systemic vasodilation [15, 16]. It has also emerged as an independent risk factor for cardiovascular collapse in critically ill patients [3]. In contrast, ketamine and etomidate generally provide greater hemodynamic stability: ketamine through its sympathomimetic properties, and etomidate through minimal cardiovascular depression, though etomidate carries a distinct limitation of adrenal suppression [17]. Remimazolam, a novel benzodiazepine, has shown a favorable hemodynamic profile in perioperative

and cardiac surgical populations [18, 19], but evidence in ICU patients is still scarce. Taken together, these observations suggest that, when cardiovascular instability is anticipated as in the critical care setting, propofol should be avoided in favor of the available alternatives, and agent selection should prioritize hemodynamic tolerance over familiarity or convenience. Hemodynamic effects of these induction agents are summarized in Table 1.

Clinical evidence

Agent selection influences outcomes. In the INTUBE study, propofol was associated with a higher risk of cardiovascular collapse regardless of dose, and was the only modifiable risk factor identified through inverse probability of treatment weighting [3]. A recent trial reported a better 7-day survival with ketamine than etomidate (85.1% vs. 77.3%), though the difference at day 28 was not statistically significant (66.8% vs. 64.1%) [20]. Systematic reviews comparing ketamine and etomidate yield conflicting results [21, 22]. One using Bayesian analysis revealed an 83.2% probability that ketamine reduced mortality [21], whereas other data suggest ketamine may increase hemodynamic instability despite lowering vasopressor use, with no survival advantage [22]. These discrepancies likely reflect methodological differences in study inclusion and statistical approaches.

Recently, a multicenter RCT of 2365 patients compared ketamine and etomidate for rapid sequence induction, showing no significant difference in 28-day mortality (28.1% in the ketamine group vs. 29.1% in the etomidate group) with a higher incidence of cardiovascular collapse with ketamine (22.1% in the ketamine group vs. 17.0% in the etomidate group; risk difference, 5.1 percentage points; 95% confidence interval, 1.9 to 8.3) and similar results in other safety outcomes [23]. Thus, the decision to use either of these drugs should be based on the clinical context, patient characteristics and local availability.

Cardiac consequences of hypoxemia and apnea

Myocardial ischemia during hypoxemia and apnea

The heart is among the organs most sensitive to changes in oxygen supply. Although coronary artery flow increases in response to hypoxemia in an attempt to maintain myocardial oxygen delivery [24], this compensatory response may be insufficient to prevent ischemia when profound hypoxemia occurs. Even brief episodes of hypoxemia can precipitate myocardial ischemia, impair contractility and further reduce cardiac output and oxygen delivery.

Peri-intubation hypoxemia is common in critically ill patients undergoing tracheal intubation, particularly among those with obesity [2, 25]. In patients with pre-existing oxygenation impairment—such as in acute respiratory distress syndrome (ARDS), pulmonary embolism, or aspiration—the risk of developing severe hypoxemia is further increased. Apnea during intubation exacerbates this risk by halting ventilation, allowing carbon dioxide (CO_2) to accumulate and pH to decline. Initially, these changes promote vasodilation and increased coronary blood flow through the direct effects of CO_2 and hydrogen ions (H^+) on vascular smooth muscle tone [26, 27]. Once pH falls below critical thresholds (e.g., < 7.2), myocardial contractility declines, adrenergic responsiveness diminishes, and arrhythmia risk rises [28]. These changes can further impair cardiac output and coronary perfusion, propagating myocardial ischemia.

In addition, sympathetic activation during hypoxemia and apnea may exacerbate arrhythmogenic risk and contribute to hemodynamic instability. Sedative agents administered during intubation may further potentiate these effects through vasodilation and myocardial depression, compounding cardiovascular vulnerability.

Right ventricular failure and hypoxemia

Right ventricular (RV) failure represents a complex and often under-recognized contributor to peri-intubation hemodynamic instability [29]. Several anatomical and physiological characteristics differentiate the right heart from the left, particularly during the peri-intubation period. First, the right coronary artery (RCA) receives blood flow during both systole and diastole, unlike the left coronary artery, which is perfused predominantly during diastole [30]. This dual-phase perfusion suggests

Table 1 Hemodynamic effects of induction agents

Drug	Cardiac output	Systemic vascular resistance	Mean arterial pressure	Heart rate	Myocardial contractility	Notes
Propofol [3, 14–16]	↓	↓↓	↓↓	↓ or →	↓	Risk factor of cardiovascular instability
Ketamine [21, 55–57]	↑	→ or ↑	→ or ↑	↑	↑	Increased cardiac oxygen demand
Etomidate [17, 58, 59]	→	→ or ↑	→	→	↓ or →	Adrenal suppression
Midazolam [60–63]	↓	↓	↓	↓ or →	↓	Delirium risk
Remimazolam [18, 19]	→	↓ or →	↓ or →	→	→	Limited data

that the right ventricle may be somewhat more tolerant of transient hypoxemia.

Second, the thinner RV wall renders it particularly susceptible to pressure overload, such as that imposed by elevated pulmonary artery pressures [31]. Third, hypoxic pulmonary vasoconstriction (HPV)—a reflexive narrowing of pulmonary arterioles in response to alveolar hypoxemia—can markedly increase pulmonary vascular resistance when large lung regions are affected, as in ARDS. In addition to HPV, pulmonary vascular obstruction by microvascular thrombi has been increasingly recognized in ARDS, further contributing to pulmonary hypertension and potential RV failure [32]. As RV afterload rises, filling of the LV falls, RCA flow diminishes, and a vicious cycle of RV ischemia and circulatory collapse may ensue [33].

Transition to positive-pressure ventilation: heart–lung interactions

The process of induction, intubation and the start of invasive mechanical ventilation represents the prototype of heart–lung interactions physiology, with changes occurring in the very short period of rapid sequence induction and involving in parallel the cardiovascular and respiratory systems. Knowledge of these phenomena may mitigate their detrimental clinical consequences [34].

Administration of hypnotic and muscle relaxant agents has an important effect on hemodynamic physiology secondary to the abolition of spontaneous breathing and the transition from negative to positive-pressure ventilation leading to abolition of an important driver of venous return and an increase of intrathoracic pressure [35]. Indeed, in spontaneously breathing patients, inspiratory effort facilitates venous return to the right atrium (leading to increased right ventricular preload), while at the end of expiration (functional residual capacity), alveolar pressure equals atmospheric pressure and, at this pressure level, pulmonary vascular resistance is at its lowest level. The abolition of negative pressure inspiration and the application of a positive pressure then reduce the pressure gradient for right atrium venous return and, in turn, this may precipitate systemic hypoperfusion [34, 35]. In parallel, the increase of lung volume may be associated with an increase of pulmonary vascular resistance, driven by the axial and radial compression of intrathoracic vessels by the alveolar unit. This, in turn, may worsen the right ventricle ejection performance [34, 36].

The hemodynamic effects of positive pressure are more profound as the mean airway pressure increases. Positive end-expiratory pressure (PEEP) is among the variables having a major influence on mean airway pressure, with a meaningful impact on cardiovascular status, especially in hypovolemic patients. Therefore, it may be safer to set the initial PEEP at a low level (e.g., 5–6 cmH_2O) and then to gradually reach its target value once the patient has been stabilized following induction and intubation. In parallel with intravascular volume and heart function optimization, awake intubation (i.e., flexible bronchoscopy intubation under topical anesthesia) has been suggested to maintain the benefit of negative inspiratory pressure on venous return and to avoid the risks of rapid induction and desaturation during apnea. This approach, albeit requiring a high level of expertise, may represent a safer option to rapid sequence induction, not only in case of anatomy but also in physiological challenges [35].

Practical considerations: pre-intubation optimization and real-time management

The physiological alterations that occur during tracheal intubation in critically ill patients often lead to marked hemodynamic instability. Preventing such deterioration requires a proactive approach to optimize cardiovascular physiology before and during intubation. The following practical strategies—ranging from preparation to post-intubation care—form the backbone of safe airway management (Fig. 2).

Preparation with a checklist and team dynamics

Careful preparation of drugs, devices, personnel, and the environment is essential to mitigate hemodynamic risks. The use of a pre-defined checklist promotes structured care and reduces errors in this high-stakes procedure [10]. However, to enhance checklists' benefit, they should include strategies specifically addressing patient's physiology and its peri-intubation optimization [37, 38]. Equally important is effective team communication, including role assignment and contingency planning. Together, structured preparation and clear communication establish the foundation for controlled intubation.

Induction agent selection

Induction agents can profoundly affect hemodynamics. Propofol is associated with cardiovascular instability regardless of dose [3]. Ketamine and etomidate offer better hemodynamic tolerance; however, their relative benefits remain uncertain. Remimazolam shows potential but lacks sufficient evaluation in the critically ill [18, 19].

Neuromuscular blockade agents (e.g., rocuronium or succinylcholine) are typically administered during induction for tracheal intubation in critically ill patients. Sedative infusion is important to avoid accidental awareness in patients who receive neuromuscular blockade agents.

Accordingly, agent selection should be individualized, prioritizing ketamine or etomidate over propofol in patients at risk. When instability is anticipated despite these choices, adjunct strategies such as volume

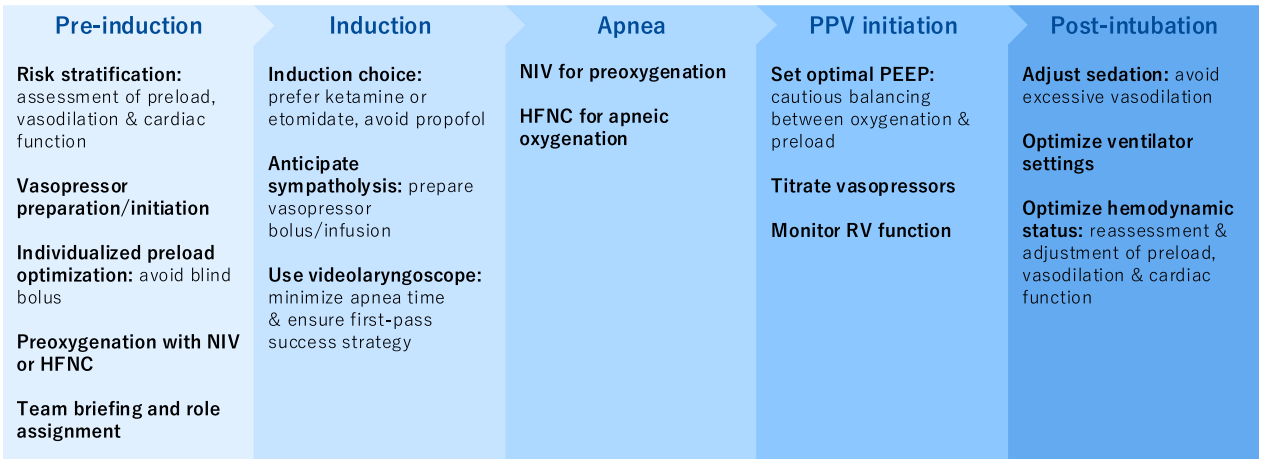
Pre-induction
Risk stratification: assessment of preload, vasodilation & cardiac function
Vasopressor preparation/initiation
Individualized preload optimization: avoid blind bolus
Preoxygenation with NIV or HFNC
Team briefing and role assignment
Induction
Induction choice: prefer ketamine or etomidate, avoid propofol
Anticipate sympatholysis: prepare vasopressor bolus/infusion
Use videolaryngoscope: minimize apnea time & ensure first-pass success strategy
Apnea
NIV for preoxygenation
HFNC for apneic oxygenation
PPV initiation
Set optimal PEEP: cautious balancing between oxygenation & preload
Titrate vasopressors
Monitor RV function
Post-intubation
Adjust sedation: avoid excessive vasodilation
Optimize ventilator settings
Optimize hemodynamic status: reassessment & adjustment of preload, vasodilation & cardiac function

Fig. 2 Peri-intubation hemodynamic care bundle in critically ill patients. While this figure focuses on physiologically difficult airway management, assessment of an anatomically difficult airway is always important for safe tracheal intubation. PPV (positive-pressure ventilation) refers to invasive mechanical ventilation after successful tracheal intubation. *NIV* (noninvasive ventilation), *HFNC* (high-flow nasal cannula), *PPV* (positive-pressure ventilation), *PEEP* (positive end-expiratory pressure), RV (right ventricle)

optimization and pre-emptive vasopressors should be considered [39]. A bundled peri-intubation approach—integrating risk stratification, team coordination, and post-intubation readiness—is essential [10].

Individualized fluid therapy

Fluid management during the peri-intubation period should be individualized to optimize preload without causing harm. A routine fluid bolus is not supported by evidence [39, 40]. Instead, clinicians should assess both fluid responsiveness (using dynamic measures, such as passive leg raising or pulse pressure variation [41, 42]) and fluid tolerance [43], the latter referring to the volume of fluid a patient can safely receive without inducing organ dysfunction.

When responsiveness is absent, fluids should be avoided; when responsiveness exists but tolerance is low, risks and benefits must be carefully balanced. Dynamic assessment of both parameters is key. Focused ultrasound may help to tailor the need of intravascular volume optimization and fluid tolerance by an assessment of both cardiovascular (e.g., ventricular and/or inferior vena cava sizes) and lung sonography (e.g., presence of a B-line pattern) [44].

Pre-emptive vasopressors and hemodynamic monitoring

Cardiovascular instability remains the most frequent peri-intubation complication [2, 39, 40]. Given the potential association of transient peri-intubation hypotension with long-term outcomes, strategies to avoid exposure to this event, instead of a reactive approach to hypotension (i.e., vasopressor administration once hypotension is observed), may be of higher benefit. Moreover, on-demand vasopressors (e.g., norepinephrine and phenylephrine) can treat hypotension but may be insufficient to prevent deterioration. Two ongoing RCTs (PREVENTION [NCT05014581] and FLUVA [NCT05318066]) are evaluating the efficacy and safety of the pre-emptive vasopressor infusion approach [45].

Arterial cannulation and invasive blood pressure monitoring should be considered before induction in all critically ill patients, given the need to prevent and promptly react to the rapid changes of the hemodynamic status the patients will experience with intubation. In a randomized single-center study investigating continuous intra-arterial vs. intermittent oscillometric arterial pressure monitoring, continuous monitoring was associated with a reduction of exposure to hypotension episodes in noncardiac surgery patients [46]. Given the higher incidence and potential detrimental effects of hypotension in critically ill patients, an arterial line should be considered before induction whenever feasible. Moreover, repeated echocardiography is also important to evaluate fluid status and left- and right-sided cardiac function. When right ventricular failure or pulmonary arterial hypertension is suspected, pulmonary arterial catheter placement may be considered for continuous monitoring of right ventricular function and optimization of hemodynamic management.

Optimizing oxygenation

Optimizing preoxygenation with appropriately selected techniques is a critical component of mitigating hypoxemia-driven cardiac complications during tracheal intubation in critically ill patients. Noninvasive ventilation (NIV), high-flow nasal cannula (HFNC), and standard

Table 2 Prioritized research questions for peri-intubation hemodynamic management

Evaluate the efficacy and safety of pre-emptive vasopressor therapy to reduce the risk of peri-intubation cardiovascular instability
Develop and validate scoring systems to predict the hemodynamic trajectory after induction
Establish guidance for early identification and management of patients with right ventricular dysfunction
Evaluate the effectiveness of a peri-intubation hemodynamic care bundle encompassing pharmacologic and non-pharmacologic strategies

facemask oxygen are the three commonly used devices for preoxygenation. NIV can provide PEEP and pressure-support ventilation, thereby relieving inspiratory efforts, increasing lung volume, lowering RV preload, and decreasing left ventricular afterload [47]. However, clinicians must also be aware that NIV imposes positive intrathoracic pressure, which may compromise venous return and systemic hemodynamics, underscoring the need for vigilant hemodynamic monitoring and tailored vasopressor support when using NIV for preoxygenation in shock or preload-sensitive patients.

HFNC can enhance oxygenation through a modest PEEP effect generated by high-flow rates and can reduce the work of breathing by flushing dead space in the upper airway [48, 49]. In addition, HFNC is often used during the apneic period—that is, during laryngoscopy—in an effort to prolong safe apnea time and attenuate desaturation risk [50].

A network meta-analysis of preoxygenation strategies in critically ill patients indicates that NIV reduces the incidence of hypoxemia compared to HFNC and facemask oxygen, particularly in patients with $PaO_2/FiO_2 < 200$ [51]. HFNC is superior to facemask oxygen [51]. No differences were observed in successful intubation on the first attempt or in mortality between preoxygenation methods [51]. Given these results, NIV should be the first-line preoxygenation method when tolerated. Apneic oxygenation using HFNC during laryngoscopy may further reduce desaturation risk and can be considered based on individual patient tolerance and institutional practice.

Choice of laryngoscopy

Videolaryngoscopy is now recommended as the first-line approach in ICU intubations. A large multicenter RCT demonstrated improved first-pass success with videolaryngoscopy compared to direct laryngoscopy [52], a finding confirmed by meta-analyses that also reported reductions in esophageal intubation and aspiration [53]. Given the importance of achieving a first-attempt intubation success in critical care, videolaryngoscopy is the elective method to prevent airway-related hemodynamic complications.

Post-intubation hemodynamic management

Transitioning to positive-pressure ventilation increases intrathoracic pressure, which can reduce venous return and left ventricular preload. Clinicians should anticipate these effects and titrate vasopressors and fluids accordingly. Given the major impact of PEEP on mean airway and intrathoracic pressures, its initial value set on the ventilator may be 5–6 cmH_2O and the target value may be gradually reached during/after patient stabilization [54]. Final adjustments to PEEP and tidal volume should aim to balance oxygenation goals with avoidance of further hemodynamic compromise. Sedatives should be carefully titrated to prevent both vasodilation and sympathetic surges. Attention to preload, vasopressor support, and ventilator settings is crucial for maintaining stability after intubation [10].

Knowledge gaps and future directions

Peri-intubation cardiovascular optimization has a high priority in the research agenda given the associated high morbidity and mortality (Table 2). Preventive interventions targeting post-induction hypotension are under investigation. Prophylactic vasopressor infusion has been proposed. Two ongoing RCTs, the PREVENTION (NCT05014581) and FLUVA (NCT05318066) trials, are explicitly designed to test this strategy. Their results are expected to clarify whether anticipatory hemodynamic support can reduce the burden of adrenergic collapse. Moreover, their results may provide important data on whether a pre-emptive approach aiming at avoiding patients' exposure to hypotension can be of benefit for long-term morbidity and mortality reduction.

Another potential question is the role of benzodiazepines in this setting. For example, it remains unclear whether co-administration of benzodiazepines (e.g., midazolam and remimazolam) with ketamine or etomidate reduces the risk of awareness during paralysis compared to adequately dosed ketamine or etomidate alone.

Conclusions

Tracheal intubation in the ICU is a hemodynamic intervention with predictable, phase-specific vulnerabilities. Baseline adrenergic dependence, abrupt sympatholysis, hypoxemia, acidosis, and adverse heart–lung interactions

act cumulatively, especially with hypovolemia, vasoplegia, or RV dysfunction, to precipitate collapse. A proactive, physiology-informed bundle (judicious induction choice, individualized fluids/vasopressors, evidence-based preoxygenation, vigilant post-intubation care) can maintain perfusion. High-quality trials of pre-emptive strategies are underway. Currently, clinical decision-making should be based on mechanistic, pathophysiological understanding with pragmatic bedside action, which will remain essential even after the evidence evolves.

Abbreviations

HFNC	High-flow nasal cannula
ICU	Intensive care unit
INTUBE	International observational study to understand the impact and best practices of airway management in critically ill patients
NIV	Noninvasive ventilation
PEEP	Positive end-expiratory pressure
RCT	Randomized controlled trial
RV	Right ventricular/right ventricle
SCCM	Society of Critical Care Medicine

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